5-2018

Enamel Hypoplasia and Its Relation to Ethnicity and Socioeconomic Status in the 19th Century United States

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ENAMEL HYPOPLASIA AND ITS RELATION TO ETHNICITY AND SOCIOECONOMIC STATUS IN THE 19TH CENTURY UNITED STATES

by

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Thesis submitted in partial fulfillment
of the requirements for the degree

of

DEPARTMENTAL HONORS

in

Anthropology
in the Department of Sociology, Social Work, and Anthropology

UTAH STATE UNIVERSITY
Logan, UT

Spring 2018

For my parents who pushed me toward college

and to my partner who kept me going while I was here.

Thank you for everything, Beeb.

Acknowledgements

I would like to thank Dr. Patricia Lambert for her instrumental input for the construction, creation, and implementation of this research project. I would also like to thank Dr. Jacob Freeman and Mckinley Benson of the Utah State University Anthropology Department for their support. Additionally, I would like to thank the Utah State University Honors and Anthropology Departments as well as the Utah State University Undergraduate Research program for providing funding opportunities and the ability to undertake this project. A big thank you to Dr. David Hunt and Naovi Dollas of the Smithsonian Institution for their kindness and curation of a wonderful collection. I would also like to give a special thanks to Adam Gifford for his continued encouragement, input, and care for our many pets in my absence.

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Abstract

Linear enamel hypoplasia (LEH) is a condition of tooth enamel characterized by linear bands in tooth enamel that result from metabolic stress during the childhood years of enamel formation. The presence of LEH has frequently been used in biological anthropology as a marker of stress experienced during childhood. This paper uses a biocultural approach to investigate the occurrence and severity of LEH defects on the teeth of African American and European American adult male remains in the Terry Anatomical Skeletal Collection. The Terry Collection consists of low socioeconomic status individuals whose remains were unclaimed at St. Louis morgues and hospitals, and thus available for inclusion in a research collection. For this research, teeth of individuals born during the Reconstruction (1865-1877) and Early Jim Crow (1877-1900) years were examined and scored for the presence and width of LEH defects. I hypothesized that 1) the rate of individuals with evidence of LEH would be high in this sample due to the low socioeconomic status of individuals comprising the collection; and that the occurrence of LEH would be similar for European Americans during both time periods, but increase for African Americans born during Early Jim Crow as a result of state sanctioned racial discrimination. Results support a high prevalence of LEH for both ethnicities and time eras with a 95% rate of occurrence. Additionally, wider LEH lesions in the African American sample indicate that prolonged stress episodes increased significantly for African Americans during Early Jim Crow, but not for European Americans. These data suggest that, while the total population experienced substantial metabolic stress during childhood, African Americans males born during the Jim Crow years experienced more prolonged periods of stress as a result of race-based discrimination.

Final Product Word Count: 5117

Introduction

The biocultural model of biological anthropology is situated in the environmental context of a population (Figure 1). Disparities in access to resources impose limitations on the biological health of individuals within an affected population. These constraints can result from cultural factors like social organization, access to technology, and access to food resources. Disparities in the ability of populations to access necessary resources can result in population-level stress (Rankin-Hill, 1997). Stress is a product of three interrelated factors: host resistance, environmental constraints, and cultural systems. These factors and their severity will vary over time and location, but if they are not mitigated they may result in physiological disruptions. Cultural systems may increase or reduce the impact of environmental constraints. Those with insufficient cultural buffering of stress can only rely on their own innate resistance, which varies by age and sex. When the innate resistance of hosts and their environmental constraints are held constant, then disparities in stress may be attributed to cultural variation (Goodman, et. al. 1984).

The purpose of this study is to address several questions related to this issue through examination of the prevalence and severity of enamel hypoplasia in a sample of individuals from the Terry Anatomical Collection, a 20th century human skeletal sample curated by the Smithsonian Institution. Specifically, I examine adult male African American and European American individuals that were born during the Reconstruction (1865 - 1877) and Early Jim Crow (1877-1900) eras. My study is designed to provide insight into the following questions: 1) does the total sample exhibit high signs of stress due to their low socioeconomic status? 2) is there a disparity in the occurrence of enamel hypoplasia between the Reconstruction and Early Jim Crow eras in individuals in the Terry Collection?; 3) are there differences between ethnic

groups in the occurrence of these growth disruptions?; and 4) does the frequency of enamel hypoplasia correlate with historical accounts of population stressors during this time?

Stress and Ethnicity

The health of an individual can be altered by factors that influence access to resources, including gender, socioeconomic class, and ethnicity. People of lower social status experience decreased access to food sources high in nutrition, as well as increased risk of disease due to poor housing conditions, inadequate nutrition, and overcrowding (Rankin-Hill, 1997; Dunnavant, 2017). When studying stress levels experienced by populations in the United States during the late 19th century, it is important to consider in particular how the codification of 'race' at that time may have affected levels of stress and access to resources for different ethnic groups, specifically for people categorized as 'black' (African American) versus those identified as 'white' (European American). These racial groupings would likely have resulted in differing levels of physiological stress in historical contexts where race-based discrimination was socially sanctioned. Therefore, as socioeconomic status shifted in consequence of a changing sociopolitical landscape, one should expect a corresponding change in the incidence of stress-related markers in those most affected by racist ideology (Nakayama, 2016).

Stress impacts on an individual's health can leave permanent, identifiable markers on the skeletal system after death. Systemic disturbances to an individual's metabolism can result in the disruption of enamel production by ameloblasts during dental development during infancy and childhood (Corruccini et. al., 1985). These childhood growth disruptions may manifest as permanent enamel defects that persist throughout the life of an individual. These defects are known to be associated with nutritional disorders as well as other pathological conditions that

cause systemic metabolic disruption during development (Cook & Buikstra, 1979; Cutress & Suckling, 1982; Davidson & Black, 2015; Kreshover, 1960; Lanphear, 1990). In order to understand what these indicators of physiological disruption mean, they must be placed in the context of a population's history and lifeways (Rankin-Hill, 1997). Historical evidence of nutritional deprivation, high stress rates, and developmental disruption should correspond with an increase in hypoplastic defects of tooth enamel (Corruccini et. al. 1985; Goodman & Rose, 1991), and the frequency and severity of these defects can therefore be used to assess rates of childhood metabolic stress in past populations (Nakayama, 2016).

Enamel Hypoplasia and Development

Enamel hypoplasia is a condition of tooth enamel characterized by deep, confluent pits or lines present in the dental enamel. It was described as early as the 1930's by Dean (1934). Today, hypoplastic defects are understood as a developmental disturbance that reduces the production of enamel by ameloblasts and thins the enamel matrix during dental development (Goodman & Rose, 1990; White & Folken, 2005). The cause of reduced enamel production has been attributed to many factors, including chronic nutritional deficiency, acute disease episodes, infectious disease, weaning, and other conditions that interfere with normal metabolism (Cook & Buikstra, 1979; Cutress and Suckling, 1982; Davidson & Black, 2015; Kreshover, 1960; Lanphear, 1990). Additionally, the size of hypoplastic defects may relate to the severity of the corresponding disruption (Suckling, 1989). When the width of enamel hypoplastic defect are considered, studies like Guita (1984) suggest that the greater band width corresponds with longer duration of a stress episode. There is also evidence that infectious diseases impacts the digestive

system's ability to absorb nutrients, causing a higher susceptibility to ameloblastic activity disruption and the development of enamel hypoplastic defects (Rose et. al. 1985).

The formation of the human dentition begins during the fifth month of gestation within the maxilla and mandible, and continues until the sixth year for deciduous (baby) teeth (Rankin-Hill, 1997). During the normal development of the tooth enamel, ameloblasts gather on the dentin-enamel junction. These ameloblasts deposit enamel at the same rate that odontoblasts deposit the inner tooth dentin. This process of enamel deposition originates on the tooth cusp and moves toward what will become the cemento-enamel junction. As a result, the tooth cusp represents the earliest tissue deposits for each individual tooth. Normally, the enamel matrix will deposit from the inside of the tooth toward the outer surface in an even layer throughout the tooth. However, metabolic disruptions to ameloblast activity causes them to lose their ability to secrete enamel, resulting in a thinner enamel deposit on the portion of the tooth forming at the time of the metabolic disruption (Goodman & Rose, 1990 & 1991).

Although the specific cause of these defects can seldom be determined, the occurrence of these defects is understood as a general indicator of metabolic stress that temporarily disrupts the function of amelogenesis (Corruccini et. al. 1985). As tooth enamel does not remodel over the lifetime of an individual, these defects can be used to document metabolic disruption during the years of dental development (Rose et al. 1985). The occurrence of enamel hypoplasia can be documented most readily and consistently on the maxillary first incisors, as well as on the mandibular canines. However, the susceptibility of ameloblasts to metabolic disruption varies within and between teeth (Rose & Goodman, 1990). This suggests that the same metabolic stress event will present differently depending on the tooth selected as well as its age at eruption. Therefore, some advocate for the analysis of all available teeth as this accounts for intertooth

variability and confirms that a defect results from systemic rather than local disruption. However, previous studies like Goodman, et. al. (1980) demonstrate that maxillary central incisors and mandibular canines account for 95% of systemic disruptions (Rankin-Hill, 1997).

As permanent tooth crowns form between the ages of about 5 months and 12 years, hypoplastic defects in adult teeth indicate that an individual experienced growth disturbances during their childhood. Additionally, because dental development follows a predictable and well-documented pattern, one can identify the approximate age that growth disruption took place from the location of a defect on a particular tooth crown, or the pattern of defects across the dentition. As a result, enamel hypoplasia can be used to indicate the age and severity of growth disruptions that resulted from stressors like poor nutrition and disease (Hassett, 2014).

Epidemiological studies of living populations have demonstrated a higher prevalence of enamel hypoplastic defects in populations experiencing malnutrition (Sweeney et al. 1969). This also corresponds with an increase in enamel hypoplastic defects in nonhuman primates during times of nutritional stress (Guatelli-Steinberg & Benderlioglu, 2006). Additionally, the sensitivity of a tooth to developing enamel defects is determined in large part by overall nutrition, with lower nutrition leading to an increased susceptibility to diseases that result in metabolic disruption (Davidson & Black, 2015). As nutrition has a significant impact on the frequency of enamel hypoplasia, the adequacy of childhood diets can directly or indirectly be determined by the presence and severity of enamel defects (Rose, et. al. 1985).

Therefore, the examination of enamel hypoplasia prevalence and severity can be used as an indicator of metabolic stress within a population. As a result, it is possible to identify an increase or decrease in population stress over time through the examination of human remains spanning several decades. Differences in the quantity and severity of dental stress markers over

time can indicate an overall change in public health or the health of certain demographics, and may show disparities that persist or diminish over time (Nakayama, 2016).

Historical Context and Inequality

Attempting to understand the past of underprivileged populations through the historical record can be exceedingly difficult, as they leave very few documents of their existence. While they were essential elements of society, providing goods, services, labor, and facilitation for the lives of the wealthy. Those of lower class were less likely than the wealthy to be documented as they did not typically create land records, personal diaries, or other written material that would have survived to this day. However, an understanding of historical inequality, in combination with skeletal data, can lend insight into the lifeways of past populations and provide an unwritten record of past social inequality (Rankin-Hill, 1997).

Inequality can be understood as an extension of structural violence that has either direct or indirect influence on the physiological and psychological health of marginalized individuals. This structural inequality manifests as a disparity in the distribution of wealth, resources, and access to medical care. The biocultural approach recognizes that structural inequalities that occurred during the life of an individual are likely to result in physiological consequences that can be seen in the bioarchaeological record (Nystrom, 2014).

The second half of the 19th century was turbulent for African Americans, as it encompassed the end of the Civil War, the emancipation of slaves during the Reconstruction era, and the establishment of Jim Crow Era segregation (Dunnavant, 2017). In the wake of the Civil War, the South was severely impacted economically, with per capita income crashing along with wages, resulting in a weakened Southern economy. Despite this, 90% of African Americans still

lived in the South from 1870-1900. Moving to Southern urban centers before the Great Migration of 1915 where large numbers of African Americans moved from the South to Northern industrial centers (Logan, 2009).

However, African Americans that migrated from rural location to urban centers created an increase in African American populations in northern as well as southern cities by nearly three times. This population expansion increased the health risk for infectious diseases like smallpox, tuberculosis, and cholera that resulted from overcrowding in these urban centers. As African Americans post-emancipation moved to urban centers in search of employment and greater freedom than was provided in rural areas, they encountered these urban illnesses and premature death (Dunnavant, 2017).

Demographers have noted a rise in mortality rates associated with an increase in industrialization in the United States during the mid-19th century. During this time, the United States population experienced rapid growth and urbanization that resulted in an increase in limited access to health care, poor sanitation, and higher risk for stress during childhood. This was particularly seen in America's cities that experienced the highest concentration of growth and urban development (Meindl & Swedlund, 1977). Logan (2009) suggests that the effects of slavery had lasting impacts on African Americans born post-emancipation, with lower standards of living and decrease in stature throughout the Reconstruction era. These impacts differentially affected those of low socioeconomic class who worked and lived in poor conditions, which exacerbated these stressors (Lanphear, 1990; Mohl & Betten 1972).

Previous research has correlated increasing sedentarism, dietary stress, population density, and infectious pathogens with an increasing prevalence of enamel hypoplasia (Goodman et al. 1980). The use of enamel hypoplasia frequency and severity can therefore provide a

measure of stress for populations living during the Reconstruction and proceeding Early Jim Crow Eras. Additionally, these defects may help to identify racial disparities in childhood health during these time frames.

My research is designed to identify and quantify the biological health impacts of these social constructs. Here I investigate factors that may have altered the health of people over time within and between contemporaneous, ethnically distinct populations. I hypothesize that the frequency of linear enamel hypoplasia will correlate with shifts in ethnic and socioeconomic status. Specifically, I test three hypotheses according to their bioarchaeological implications as outlined below:

H_1: As Jim Crow laws reduced opportunities and access to resources, physiological stress increased in children of African American descent, especially those living in regions where these laws were most prominent and heavily enforced. If the health experience of African Americans did decline with increasing social circumscription, then the rate and severity of hypoplastic defects will increase over time for African Americans.

H_2: As Jim Crow laws did not impact European Americans access to resources, they would not have experienced the same severity of stress as their African American counterparts. As European American health was not impacted by racial laws, a change in the severity of enamel hypoplastic defects will not be apparent in the teeth of contemporaneous European Americans..

H_3: The Terry Anatomical Collection is comprised of people who were unclaimed at St. Louis morgues and hospitals and used as medical cadavers in the early 20th century. Therefore, this sample most likely represents individuals of low socioeconomic status without access to proper nutrition and healthcare. As a result, there will be a higher prevalence of enamel

hypoplasia overall in the Terry Collection sample than in other human skeletal samples comprising a broader representation of their respective populations.

Terry Collection Background

The Robert J. Terry Anatomical collection consists of the skeletal remains of people born between 1828 and 1943, and is primarily composed of unclaimed individuals obtained from St. Louis hospitals and morgues during the early 20th century. It was common practice during that time to use unclaimed bodies for scientific research and as cadavers in medical schools. As a result, the sample predominantly comprises individuals of low socioeconomic status who did not have the means to pay for proper burial (Hunt & Albanese, 2005), and therefore those within this collection can be seen as having suffered from structural inequality both in life and in death. Therefore, this collection is comprised of individuals of low socioeconomic status and therefore more subject to stressors that would have resulted in physiological consequences than the general population of the same time and place (Nystrom, 2014).

All individuals in the collection retain morgue records that provide year of death, race, sex, and cause of death. It is important to note that the racial assignment for these individuals was based on perceptions of race at the time, in most cases those of hospital and morgue personnel (1917-1966). Therefore, while an individual may have been identified as African American or European American, this categorization was not based on known ancestry or even on self-identification in most cases, interpretation of what characteristics assigned an individual to a racial category would have shifted over time (Hunt & Albanese, 2005). In this study I make the assumption that the racial assignments given to these individuals at death were based on the superficial characteristics such as skin color that tend to define race in the U.S., and

correspondingly would have impacted how they were perceived and their experiences and opportunities during life.

The sample used for this study consists of male adults born during the Reconstruction (1865 - 1877) and Early Jim Crow (1877-1900) eras identified as African American and European American at the time of death. The age at death is significantly younger for individuals born during the Early Jim Crow era as a result of when skeletal collection began. In the first decade of the 20th century, when the Terry Collection was initially established, it was primarily comprised of male adults of both races. As a result of collecting individuals who had died in the early 20th century, those born during the Reconstruction Era were necessarily older than those born during Early Jim Crow (Table 1). Therefore, the difference in ages for each sample results from collection methods, rather than an early age at death for the population represented (Hunt & Albanese, 2005). Thus, the comparison necessarily involves older men from the Reconstruction Era with younger men from the Early Jim Crow years.

Materials and Methods

In this study I use the frequency and width of linear hypoplastic defects as indicators of childhood health stress to examine health disparities between and among ethnically and socioeconomically distinct subpopulations in the United States, specifically between African American and European Americans born during the Reconstruction (1865-1877) and Early Jim Crow (1877-1900) eras. This study uses the generalized model for the biocultural analysis of skeletal remains developed by Goodman, et. al. (1984) to understand how environmental constraints and culturally induced stressors can cause physiological disruptions to hosts in the form of growth disruptions and disease.

The total sample consisted of 102 adult male skeletons from the Terry Anatomical Collection. The Reconstruction era sample consisted of 26 African Americans and 26 European Americans with the Early Jim Crow era sample consisted of 29 African Americans and 24 European Americans (Table 1). The age structure of the sample skews toward older individuals in the Reconstruction era subset, with a mean age of 64.25, and is significantly younger in the Early Jim Crow era sample with a mean age of 36.57 as noted (Table 2, 3, & 4). As a result, the sample is not an accurate representation of the age-at-death profile for these populations.

Previous studies note that hypoplastic defects are most readily found on the mandibular canine and central maxillary incisors, which account for 95% of total growth disruption in an individual (Goodman et. al., 1980; Lanphear, 1990). Goodman and co-authors (1980) suggest that no information is lost if only canines and incisors are used in measurement of overall enamel hypoplasia occurrence in an individual. Canines alone provide the best records for stress from 3 to 6.5 years of age (Goodman et. al. 1980). For this study observations were made for all available canines with at total possible of 4 and incisors with a total possible of 8. Individuals were included in the sample if they retained at least one of these teeth. This sample was then broken down into maxillary and mandibular incisors and canines.

While hypoplastic defects of tooth enamel under examination for this study formed in childhood and were present throughout the life of the individuals in the sample, the number and completeness of teeth varied depending on the age of individuals at death. Those born during the Reconstruction era were more often older and retained fewer teeth that could be scored for enamel hypoplasia than those born during Early Jim Crow era. That said, all individuals in the sample were equally subject to post-mortem attrition due to many years of handling while in the

collection. As a result, the number of individuals with scorable teeth was severely limited in comparison to the overall sample size .

For the purposes of this study, teeth with less than 25% of observable surface due to wear, damage, or calculus buildup that prevented observation of the tooth enamel were eliminated from consideration. The methodology for diagnosing enamel hypoplasia built upon Dean (1934) and Goodman and colleagues (1980) by defining a hypoplastic defect as an observable, transverse area of depressed enamel. The type of linear defect was categorized as striations representing linear enamel defects (LEH), which occurred most commonly, or as interrupted, linear pitting (PIT). The position of the defect was located using a 10x powered magnifying lamp and defects were measured from the cemento-enamel junction to the bottom edge of the defect using needle-nose, Mitutoyo, sliding digital calipers calibrated to 0.00 mm (Corruccini et. al. 1985). Additionally, the width of the defect was measured from the bottom edge to the top edge to the nearest .01 mm. Results from this study were recorded and depicted graphically on a worksheet (Figure 2) for each individual and then entered into SPSS for statistical analysis. An independent samples t-test was used to compare measurements of greatest defect width for each tooth types. The number of teeth retained and the number impacted with enamel hypoplasia were generated to create a present of teeth impacted for each tooth types and each time and ethnic category.

Results

The sample consisted of 102 individuals of known date of birth with 1 individual having an unknown date of death. Of these individuals, 55 were identified as African American and 47 as European American. There were 49 individuals, 26 of which were African American, born

during the Reconstruction era. The remaining 53 individuals, 29 of which were African American, were born during Early Jim Crow (Table 1 & Figure 3).

Of these individuals the median age at death was 64.25 years for those born during the Reconstruction era and 36.57 years for those born during the Early Jim Crow years (Table 2). The average age at death for the total sample of African Americans was 48.51, 2.66 years younger than European Americans in the sample (Table 3). However, this lower lifespan for African Americans was only found in the Early Jim Crow sample, with a mean age of 39.82 for African Americans, 5.97 years younger than European Americans. In contrast, African Americans born during Reconstruction era had a mean age of 64.85, 1.3 years older on average than European Americans born at the same time (Table 4).

Enamel hypoplasia was present in almost all individuals in the sample, with a 94% rate of occurrence overall (Table 5). Enamel hypoplasia occurred at a rate of 88.56% for African Americans and 95.65% for European Americans in the Reconstruction sample, while both African Americans and European Americans in the Early Jim Crow sample had an enamel hypoplasia rate of 96%. There were no significant differences in the occurrence of enamel hypoplasia between ethnicities or for the different eras.

When comparing the mean number of defects per tooth for each tooth types during each era for both ethnicities, the highest mean number of defects was observed on the canines of European Americans born during the Early Jim Crow years (n=2.05). This was followed by 1.65 defects in the maxillary incisors of African Americans born during the Reconstruction era, and 1.08 for the mandibular incisors of both African Americans and European Americans born during the Reconstruction era (Table 8).

Using an independent samples t-test, comparing African American's between Reconstruction and Early Jim Crow, maxillary incisor defect width neared significance (t=-1.927 p=.061) as well as mandibular canine width (t=-1.802, p=.078). No other comparisons neared significance at (p=.050) (Table 7).

Discussion

Despite both ethnicities in both eras having a high occurrence of enamel hypoplasia (Table 5), when hypoplastic defect width was compared using an independent samples t-test, no statistically significant differences occurred. This was also true for the number of defect bands per tooth for all tooth types (Table 8). However, while these were not statistically significant results, there was a trend toward a higher quantity and larger width of enamel hypoplasia defects on the maxillary incisor of African Americans born during Early Jim crow (Table 7 & 8, Figure 3). This indicates that African Americans born during Early Jim Crow experienced more severe periods of childhood stress compared to African Americans born during the Reconstruction. Additionally, African Americans born during Early Jim Crow had greater defect widths than those of European Americans born at the same time. These was not as evident in the defect widths between European Americans and African Americans during the Reconstruction era. Additionally, European Americans born in both eras did not experience as much of an increase in defect width over time as African Americans (Table 7). This indicates that European Americans born during Early Jim Crow did not experience more severe periods of childhood stress compared to European Americans born during Reconstruction. In contrast, African Americans born during Early Jim Crow did see a significant increase in more very wide enamel hypoplastic defects than African Americans born during the Reconstruction.

However, when we break this down to tooth type and the actual width of hypoplastic defects we find that during Early Jim Crow, maxillary incisor defect width for African Americans was very similar to mandibular canine hypoplastic defect width for European Americans or the same time era. However, the sample for African Americans was larger at 27 individuals against 20 European Americans (Table 6). As a result of disparities in sample sizes and age distributions, the differences in the width and occurrence rates of enamel hypoplastic defects are largely mitigated. However, it is still of note that this sample has an extremely high overall occurrence of hypoplastic defects that are indicative of severe childhood stress for individuals of low-socioeconomic status.

High very wide enamel hypoplastic defect occurrence may be indicative of higher stress associated with malnutrition and disease. The higher occurrence of very wide enamel hypoplastic defects for African Americans born during the Early Jim Crow era indicates more severe or prolonged stress periods. This shows the biological impacts of racial discrimination that occurred during this era. Additionally, while both ethnicities had evidence of enamel hypoplasia that may be indicative of low socioeconomic status, African Americans experienced greater stress that is likely related to institutionalized racial discrimination that occurred during Jim Crow. This indicates that social constructs like race can cause greater markers of stress on the dental anatomy of individuals within a subjugated class.

Epidemiological studies of contemporary communities suggest that the occurrence of enamel hypoplasia was more frequent in individuals from underdeveloped countries. With enamel hypoplasia typically occurring at a rate of 10% or less in populations from developed countries. Additionally, those with enamel hypoplasia tended to be short for their age with families of low quality living conditions. This suggests that those with a higher prevalence of

enamel hypoplasia are more likely to have lived in poor communities with limited access to resources (Goodman & Rose, 1991[1]).

In an industrial slave cemetery consisting of 31 adults from Frederick County, Maryland representing the late 1790s - 1820's, there was a 71% occurrence of enamel hypoplasia in males with 43% in females, suggesting high rates of infection and/or nutritional deficiency (Kelley & Angel, 1983). Another slave cemetery sample just outside of Charleston, South Carolina consisted of 36 individuals, 28 adults (13 male and 15 female) as well as 8 subadults interred between 1840 - 1870. This study found an incidence of enamel hypoplasia in 92% of males and 70% of females (Rathbun, 1987). Additionally, a study of Newton Plantation slaves in Barbados that consisted of 103 individuals found a 60.7% occurrence of severe linear enamel hypoplastic defects that they defined as being major growth arrests (Corruccini, et. al. 1985).

The First African Baptist Church cemetery site in Philadelphia consisted of free African Americans born in the 19th century and contained 50 adults with scorable dentition. Of those the authors found enamel hypoplasia presence at rate of 84.3% with two or more defects. Of these defects, 69.2% occurred on the mandibular canines with 64.1% on the maxillary incisors and 37.1% on the mandibular incisors. This study attributes the occurrence of enamel defects to weaning stress as the position of the defect on the tooth shows that growth disruptions began at 2.0 years and then peaked between 3.0 - 3.5 years of age. However, they suggest that a high occurrence of diarrheal diseases due to contaminated water in Philadelphia would have contributed to enamel hypoplasia presence. The majority of those with enamel hypoplasia had growth disruptions that happened prenatally at 84.3% rate of occurrence. This suggests that childhood as well as maternal environment experienced stress episodes (Rankin-Hill, 1997).

In contrast to the African American burials discussed above, the Weir Cemetery from Manassas, Virginia, consisted of 13 individuals from a wealthy European American plantation family interred from 1841 – 1907. This sample had a lower rate of 53.8% occurrence of enamel hypoplasia. Evan in the Oneida County Poorhouse population sample of 39 poor rural European Americans interned from 1880 – 1894, a rate of occurane of 61.5% was much lower than the Terry Collection males (Little, et. al., 1992).

These studies suggest that, while enamel hypoplasia occurrence was frequent throughout America in the 19th century, the Terry Anatomical Collection consists of individuals with an unusually high rate of enamel hypoplasia. This is only rivaled by the Charleston, South Carolina slave cemetery with a 92% rate of hypoplasia occurrence in males (Rathbun, 1987). This suggests that, like the enslaved of Charleston, the poor and disadvantaged gathered for the Terry Collection experienced high rates of childhood stress that likely carried on throughout their lifetime, ending in their ultimate utilization as medical school cadavers.

Conclusion

This study has provided quantifiable physical evidence for the deleterious health impacts of social marginalization in the 19th century United States. This project contributes to the growing body of literature on the complex factors that contribute to the formation of enamel hypoplasia (Cucina et al., 1997); Nakayama, 2016; Robb et al., 2001), and how it relates to structural systems of inequality. This research sheds light on how socioeconomic as well as racial disparities impact childhood health and how these impacts have changed over time in America. More specifically, it suggests that while there are differences in the severity of childhood stress along racial lines in late 19th century United States, the major evidence for

childhood stress relates to long term inequality for the poor and segregated (either by race or class) in 19th century urban centers.

Through this small documentation of how social demographics affected the biological health of children in this country, this study demonstrates that socially derived disparities in access to and control over resources can have physiological impacts that can result in skeletal consequences. There is value in viewing bioarchaeological data as a way to facilitates communication between sociocultural and biological anthropology while promoting anthropological engagement with structural inequality. This is essential, as the structural inequality is that it is deeply intertwined within past and present political and economic organization becomes normalized if not put into this context (Farmer et al., 2006; Nystrom, 2014)

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Authors Biography

Amanda Cook was raised in the very small town of Garden City, UT overlooking the turquoise Bear Lake. During high school she strived to stand out academically, attaining honors as well as an associate degree from Utah State University by graduation. Amanda choose anthropology as her major in high school and during her college career she has fully embraced the field and gotten close to her professors along the way. During her time at Utah State, Amanda was an Undergraduate Teaching Fellow for two years, attended a Spanish study abroad in Logrono, Spain, participated in the 2016 archaeology field school, and worked on two research projects. Her research project titled, "Be Fruitful and Multiply: Fertility and Tradeoffs in Latter Day Saints" was presented at the American Association of Physical Anthropologists annual conference in New, Orleans. Additionally, Amanda designed the exhibit "Forensic Anthropology" on display in the USU Museum of Anthropology. She will be graduating with honors in the spring of 2018 with her BA in Anthropology, minor in biology, and a museum studies certificate. Amanda will an archaeology intern for the Bureau of Land Management the summer after graduation and will be applying for graduate schools in the fall. She hopes to present her current research at the American Association of Physical Anthropologists conference in 2019.

Reflection Total Word Count: 1007

Reflection

This research project gave me the opportunity to advance my undergraduate career in biological anthropology through the hands-on examination, documentation, and research of human skeletal remains. As a result of this project, I gained experience applying for funding and arranged to conduct research at an institution other than my own. While this experience was challenging, I believe that it will be valuable for future research that I may conduct in graduate school and further on in my educational career. I was able also to use this opportunity to further develop my understanding of statistical analysis and how it may be applied in the understanding of past population dynamics. More specifically, this research has provided an me with an avenue for understanding how structural inequality can produce skeletal manifestations on human remains that are indicative of reduced access to resources like health care and adequate nutrition. I believe that this capstone project has helped to substantially shape my interests and scope of research that I would like to investigate in the future as I continue my education.

As a part of conducting this research, I was afforded the opportunity to develop a relationship with my mentor that has been extremely valuable by enabling me to strengthen my interests. By working with a professor who is both exemplary in my program as well as the field of biological anthropology, I was able to gain insight into what it takes to be a successful professional in my field. By working with Dr. Lambert, I deeply expanded my understanding of the field of biological anthropology while also developing a deeper appreciation for the intricacies of research on past human behavior. Dr. Lambert provided valuable insight and perspectives that enabled me to produce a meaningful and valuable project that I believe to be relevant to the greater scientific community. I initially asked Dr. Lambert to be my mentor as I

found both her field of interest and that classes that I had the opportunity to take from her to be incredibly inspiring. After having a chance to work more thoroughly with her, I have only gained a greater appreciation for her work and for what she provides for my department. I am very lucky that she was willing to take the time to help me create and succeed in this project.

While Dr. Lambert was extremely helpful with this project, I still ran into several difficulties along the way. The biggest of which being an overall lack of time to complete this project in a way that would have left me satisfied. As a result of my other work, class, and honors obligations, I did not feel like I was able to give this project the time that it deserved. While this may be disappointing, it has inspired me to continue on with this project in the future. In addition to a lack of time, I felt inherently disadvantaged due to my difficulties in understanding statistics. Dr. Lambert was able to help me with this deficiency, but I still have hard time wrapping my mind around the topic. As a result, I believe that further mentoring on the subject would have been helpful in successfully testing my results without as much difficulty as I experienced. If I were to do this project over again, I would have given myself more time and taken a statistics course before beginning research.

One of the most valuable aspects of this project was the ability to expand my research interests within and outside of my major. As a part of this project, I conducted extensive research on topics in biological anthropology as well as fields that I had not considered relevant before. In order to evaluate how enamel hypoplasia can be used to understand past social inequality, I had to both understand the historical context of the time frame individuals were born, as well as the physical mechanics of enamel hypoplasia development. As a result, I strengthened my understanding of how biological anthropology uses enamel hypoplasia in a bioarchaeological context through thorough examination of the relevant literature. In addition to this, I examined

the dental medical science view of enamel hypoplasia formation and a historical perspective of inequality that occurred during time periods I studied. Through the study of works from all of these disciplines, I was able to gain a broader understanding of the multifaceted factors that resulted in the data I was able to gather from the skeletal remains of the Terry Anatomical collection. It is my belief that this interdisciplinary approach was essential in understanding the broad contexts that interplay in the use of dental enamel hypoplasia as a measure for past population stress.

I believe that this project is of great use to the global community as it enables a way to understand how social and culture factors alter the health of children and how this in turn impacts their overall quality of life as adults. As the people I studied had a high rate of enamel hypoplasia and were unclaimed at the time of death, it is likely that they were both born and died in harsh conditions. I believe that this demonstrates that during the late 19th to early 20th century, individuals of low socioeconomic status faced great difficulty in improving their quality of life over time. It is my belief that this research enabled a greater understanding of the factors that limit upward mobility in groups that are socially marginalized. This is essential, as it may create better methods for understanding how socially created inequality can have lasting effects as well as result in inequalities in physical health. By conducting this research, I have broadened my scope and understanding of how biological anthropology can work to address structural inequality and how this field may be used to provide deeper context for problems that persist to today. It is my hope that by understanding the interplay of factors that create inequality, we may be able to create solutions for the systemic inequality very much alive and present in today's society.

Tables and Figures

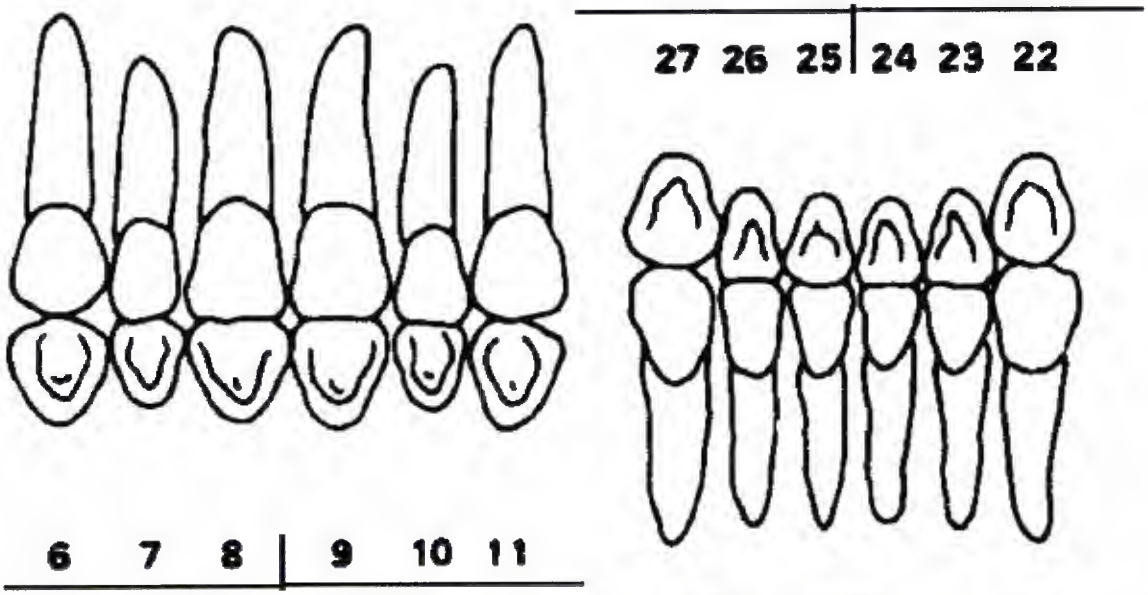
Figure 2: Dental Inventory Sheet

Dental Analysis & Visual Recording Form: Permanent Dentition

Catkey/Terry Number _____/_____ Date _____

YOB _____ Age _____ Race _____ Pic (Y/N) _____

Right **Left**



		Maxillary						Mandibular					
		Right			Left			Right			Left		
		C	I2	I1	I1	I2	C	C	I2	I1	I1	I2	C
Dental Health	Type												
	%TCP												
	Tooth L												
Enamel Hypoplasia	D to EH												
	WHL												
	D to EH												
	WHL												
	D to EH												
	WHL												

Codes

Type: 0 = Tooth Absent/Unscorable 1 = no hypoplasia 2 = linear horizontal grooves 3 = linear vertical grooves 4 = linear horizontal pits 5 = nonlinear arrays of pits 6 = single pits

%TCP Percent of Tooth Crown Present: 0 25 50 75 100

Figure 3: Year of birth and age at death for each age category.

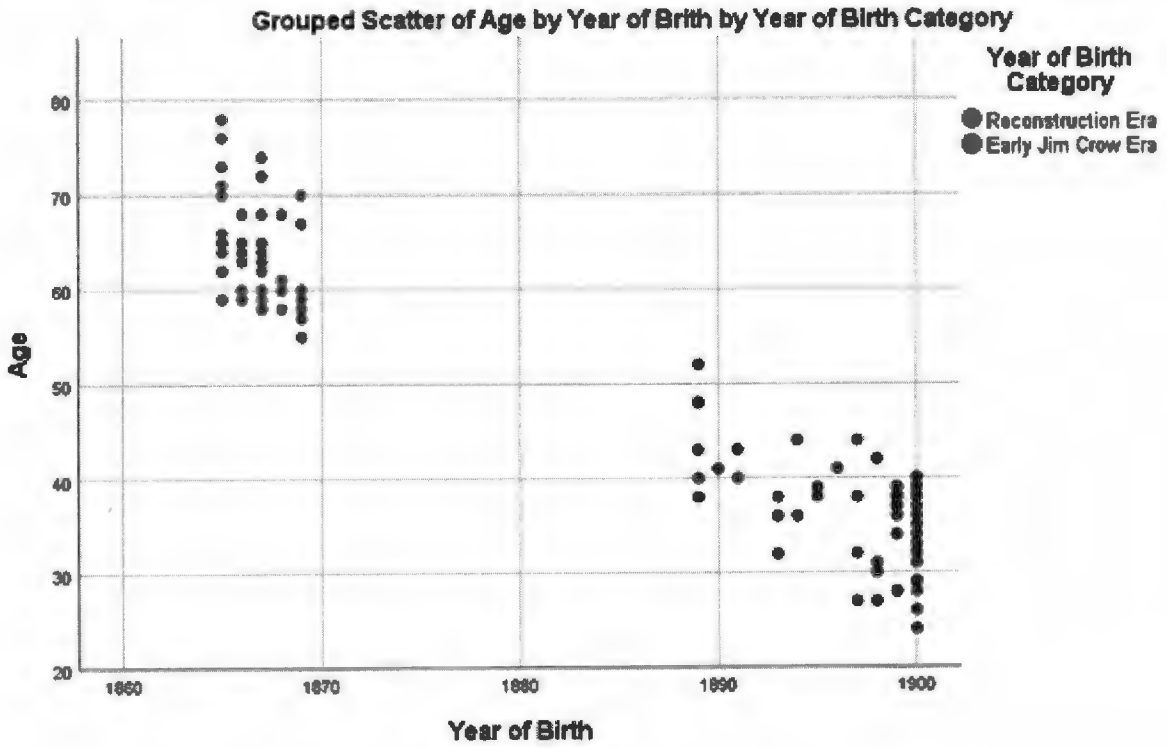


Figure 4: Widest EHP maxillary incisor defect for African Americans between Reconstruction and Early Jim Crow (t=-1.927, p=.061).

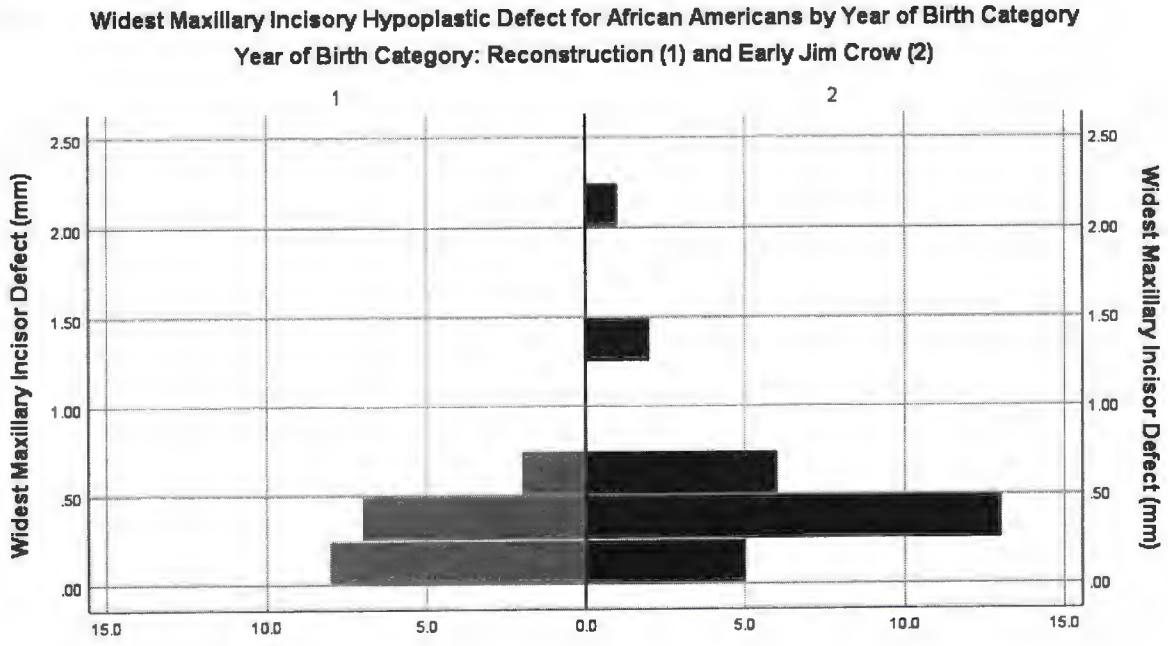


Table 1: Sample breakdown

	African Americans	European Americans	**Total**
Reconstruction Era	26	26	49
Early Jim Crow Era	29	24	53
Total	55	47	102

Table 2: Age at death for individuals born during Reconstruction and Early Jim Crow eras.

Year of Birth Era	N	Mean	Std. Deviation
Reconstruction	48	64.3	5.60
Early Jim Crow	53	36.6	5.91
Total	101	49.7	15.03

Table 3: Age at death for African American and European American individuals.

Ethnicity	N	Mean	Std. Deviation
African American	55	48.5	16.42
European American	46	51.2	13.22
Total	101	49.7	15.03

Table 4: Age at death for African American and European American individuals in each era.

Ethnicity	Time Period	N	Mean Age at Death	Std. Deviation
African American	Reconstruction Era	26	64.6	5.42
	Early Jim Crow Era	29	33.9	4.87
European American	Reconstruction Era	23	63.55	5.86
	Early Jim Crow Era	24	39.83	5.45
	Total	**102**	**49.7**	**5.40**

Table 5: The prevalence of linear enamel hypoplasia in both ethnicities for each time period.

Ethnicity	Time Period	N	Mean Number of Teeth	Std. Deviation	Mean Number Hypoplastic Teeth /Person	Std. Deviation	Percent of people with Hypoplasia
African American	Reconstruction Era	26	5.7	3.17	3.7	2.71	88.6%
	Early Jim Crow Era	29	9.7	2.17	6.8	2.64	96.6%
European American	Reconstruction Era	23	5	3.02	3.9	2.66	95.6%
	Early Jim Crow Era	24	6.1	2.77	4	2.83	95.8%
	Total	**102**	**6.77**	**3.327**	**4.7**	**2.91**	**94%**

Table 6: Total quantity of each tooth type and percent affected for each time period and ethnicity.

Ethnicity	Time Period	N	Mean Max Incisors Per Person	Std. Deviation	Mean % Max Incisor w/ EHP Per Person	Std. Deviation
African American	Reconstruction Era	17	2.41	0.939	69.59%	0.442
	Early Jim Crow Era	27	3.04	0.854	63.93%	0.367
European American	Reconstruction Era	13	1.85	1.068	77%	0.37
	Early Jim Crow Era	14	2.14	1.406	58.93%	0.496
		N	Mean Man Incisors Per Person	Std. Deviation	Mean % Man Incisor w/ EHP Per Person	Std. Deviation
African American	Reconstruction Era	20	2.75	1.293	64.20%	0.415
	Early Jim Crow Era	28	3.36	1.062	69.64%	0.393
European American	Reconstruction Era	14	2.62	1.193	64.86%	0.463
	Early Jim Crow Era	20	3.25	0.786	42.10%	0.419
		N	Mean Max Canine Per Person	Std. Deviation	Mean % Max Canine w/ EHP Per Person	Std. Deviation
African American	Reconstruction Era	15	1.4	0.507	60%	0.471
	Early Jim Crow Era	28	1.82	0.39	60.71%	0.459
European American	Reconstruction Era	15	1.33	0.488	86.67%	0.352
	Early Jim Crow Era	15	1.27	0.458	86.67%	0.352
		N	Mean Man Canine Per Person	Std. Deviation	Mean % Man Canine w/ EHP Per Person	Std. Deviation
African American	Reconstruction Era	20	1.6	0.503	67.50%	0.467
	Early Jim Crow Era	28	1.82	0.39	80.36%	0.343
European American	Reconstruction Era	23	1.52	0.511	84.78%	0.317
	Early Jim Crow Era	20	1.7	0.47	82.50%	0.335

Table 7: Mean hypoplastic defect width for tooth types as well as time period and ethnicity.

Ethnicity	Time Period	N	Mean Max Incisor Defect Width Per Person	Std. Deviation
African American	Reconstruction Era	17	**0.277**	0.195
	Early Jim Crow Era	27	**0.51**	0.472
European American	Reconstruction Era	13	0.385	0.296
	Early Jim Crow Era	14	0.271	0.267
		N	Mean Man Incisor Defect Width Per Person	Std. Deviation
African American	Reconstruction Era	20	0.242	0.158
	Early Jim Crow Era	28	0.326	0.215
European American	Reconstruction Era	14	0.279	0.274
	Early Jim Crow Era	20	0.258	0.359
		N	Mean Canine Defect Width Per Person	Std. Deviation
African American	Reconstruction Era	22	0.373	0.270
	Early Jim Crow Era	28	0.498	0.218
European American	Reconstruction Era	23	0.537	0.331
	Early Jim Crow Era	22	0.526	0.328

Using an independent samples t-test, comparing African American's between Reconstruction and Early Jim Crow maxillary incisor defect width neared significance (t=-1.927 p=.061) mandibular canine width (t=-1.802, p=.078). No other comparisons neared significance at (p=.050).

Table 8: Mean hypoplastic defect band quantity for tooth types as well as time period and ethnicity.

Ethnicity	Time Period	N	Mean Max Incisor Defect # Per Person	Std. Deviation
African American	Reconstruction Era	17	1.65	1.455
	Early Jim Crow Era	27	1.44	0.847
European American	Reconstruction Era	13	1.15	0.689
	Early Jim Crow Era	14	0.93	0.917
		N	Mean Man Incisor Defect # Per Person	Std. Deviation
African American	Reconstruction Era	19	1.08	1.004
	Early Jim Crow Era	28	1	0.609
European American	Reconstruction Era	13	1.08	0.76
	Early Jim Crow Era	20	0.85	0.933
		N	Mean Canine Defect # Per Person	Std. Deviation
African American	Reconstruction Era	21	1.76	1.221
	Early Jim Crow Era	28	1.6	0.922
European American	Reconstruction Era	23	2	0.905
	Early Jim Crow Era	21	2.05	0.865